Angiogenesis is an important prognostic factor in ovarian carcinoma (OC) and other Mullerian tract cancers (MTCs) such as fallopian tube cancer (FTC), peritoneal papillary-serous carcinoma (PPSC), and other Mullerian adenocarcinomas. A variety of angiogenesis inhibitors (e.g., VEGF) plays a crucial role in tumor angiogenesis related to MTCs. Bevacizumab (Bev) is a humanized monoclonal antibody (mAb) that is highly selective for the soluble subunit of the vascular endothelial growth factor receptor (VEGFR). Recent studies have shown that Bev is approved for the treatment of primary advanced stage OC, FTC, and PPSC in addition to platinum-based chemotherapy (CTx). Most recently, Bev has been demonstrated to add substantial activity to conventional CTx in randomized trials in both platinum-sensitive and platinum-resistant relapsed MTCs. In platinum-refractory OC, Bev can be regarded as active as a single-chemotherapeutic agent used in this setting. Bev has also been combined successfully with mTCTs such as the docetaxel or cyclophosphamide (CPA). Nonetheless, the role of Bev in pretreated MTCs has not been defined in a manner that limited clinical experience exists so far showing the optimal regimen for this drug has to be used. This paper presents a retrospective analysis Bev-based salvage therapy in patients (pts) with heavily pretreated OC, FTC, PPSC, and MTC.

**RESULTS**

Adverse reactions associated with Bev based Tx were hypertension, proteinuria, infection, asthenia, and gastrointestinal (G1-G3). Hematologic side effects include neutropenia, anemia, or thrombocytopenia, were mainly attributable to simultaneously administered CTx as also observed in the BEVACIZUMAB DURING THERAPY 4.0 scale. Response to Tx was determined by using the RECIST 1.0 criteria and evaluated by RECIST 1.1 in all other measurable measurable lesions. In pts evaluating with disease progression only response to Bev was reported in regard to the Bevacizumab (G1-G3). Bev was administered at either 10 mg/kg IV q3w or 15 mg/kg q4w. Bev toxicity was recorded from the start of Bev based Tx until last observation or death. OS was calculated from the start of Bev based Tx until death of any cause or loss to follow-up.

**CONCLUSIONS**

Bevacizumab-based salvage therapy was feasible in patients with heavily pretreated advanced epithelial ovarian and other Mullerian cancers such as fallopian tube cancer, peritoneal papillary-serous carcinoma, and type II endometrial cancer.

In the treated population of patients, bevacizumab-based therapy was generally well tolerated.

Toxicity was manageable even in relatively frail patients with a low initial performance status.

Bevacizumab-related side effects were not therapy-limiting with very few exceptions. In particular, intestinal perforations were not observed in this group of patients while being on therapy despite their intensive pretreatment.

Bevacizumab-based salvage therapy was effective in patients with heavily pretreated patients with epithelial ovarian cancer and other advanced Mullerian cancers.

Clinical platinum-resistance did not result in an impaired likelihood of both response and survival.

Combinations of bevacizumab and conventional chemotherapy did not offer any advantages over bevacizumab monotherapy or bevacizumab-based metronomic therapy in the patient population studied.

Due to the manageable toxicity profile, bevacizumab-based therapy can be given to relatively frail patients although they have a significantly poorer chance to experience long-term responses.

Bevacizumab-based therapy appears to be a valuable option for the salvage therapy of heavily pretreated patients with ovarian cancer and other Mullerian carcinomas irrespective of their clinical platinum resistance status.

When used as salvage therapy in heavily pretreated patients, bevacizumab should be preferably given as monotherapy or combined with metronomic therapy.